



Asbestos: Facts and Fiction

I appreciate the opportunity to address the omissions, inaccuracies, and innuendos in the position paper by David P. Rall, "Media and Science: Harmless Dioxin, Benign CFCs, and Good Asbestos" (*EHP* 102:10). Unfortunately, Rall not only misinterprets the conclusions of our 1990 *Science* article by Mossman et al. (1) but he fails to mention the database, scientific panel reports, and working groups which have supported our views.

Our 1990 article reported on recent papers in the peer-reviewed literature and two international symposia, one at the International Agency for Research on Cancer (2) and the other at Harvard University (3), all concluding that chrysotile fibers are less active than amphibole types (crocidolite, amosite, tremolite) of asbestos in the causation of mesothelioma in man. In his summary of the IARC meeting, Sir Richard Doll, an eminent epidemiologist, concluded "there is the difference between the effects of chrysotile and amphiboles, which is so great in relation to mesothelioma that it is possible to argue that chrysotile does not cause mesothelioma at all" (2). This observation has been supported by numerous peer-reviewed papers and working groups subsequently (4-6).

Rall's statement that "countervailing human data on the carcinogenic effects of chrysotile asbestos (including large numbers of mesotheliomas among Canadians)" exist is reminiscent of a similar claim by Nicholson et al. (7) in which his exaggerated numbers were correctly put into perspective by the epidemiologists studying the Canadian workers (8). His unreferenced conclusion that mesotheliomas are "largely from chrysotile exposure" in insulation workers and family members who were exposed to "low doses" ignores the fact that these individuals encountered mixed exposures to chrysotile and amphiboles at much higher concentrations than levels of asbestos (predominantly chrysotile) occurring in homes and public buildings today. Moreover, Rall does not acknowledge the significant content of amphibole fibers in the lungs of these workers (9) as well as recent studies showing a correlation between the lung burden of tremolite, but not chrysotile, in the lungs of Canadian miners with mesothelioma (10).

In stating that "a threshold of effect has never been found" for asbestos, Rall stands

behind the outdated "one fiber can kill" theory of carcinogenesis. However, Rall fails to mention data supporting a threshold for chrysotile in lung cancer (11, asbestosis (12), and mesothelioma (13) as well as a panel report from the Health Effects Institute-Asbestos Research (HEI-AR) detailing animal and *in vitro* dose-response studies exhibiting no-observed adverse effect levels (NOAELs) for asbestos (14). Our recent work documents a dose-dependent increase in asbestos-induced proto-oncogene activation in mesothelial cells with no induction at lowest concentrations of fibers tested and an enhanced potency of crocidolite asbestos in comparison to chrysotile (15).

The emerging database indicating: 1) extremely low concentrations of airborne asbestos fibers in public buildings, schools, and outdoor air as compared to past levels giving rise to disease; 2) minuscule risks from asbestos at levels in indoor and outdoor air today when contrasted with other voluntary and involuntary risks in modern society; and 3) protracted, higher airborne concentrations of fibers after improperly performed asbestos removal operations, led us and other (14,16-18) to question the often unmerited and financially devastating consequences of asbestos abatement from schools and public buildings as well as possible dangers to asbestos-removal workers. As quoted in a recent council report from the American Medical Association (16):

Several editorials and scientific articles have attempted to instill a sense of reason into a debate that focuses primarily on a misunderstanding of health risk . . . Physicians and others in medicine and biology, on the other hand, must continue to drive home to the public the far greater causes of morbidity and mortality, such as smoking, drug and alcohol abuse, improper diet, and inadequate exercise.

Rall's assertions that the arguments made by the authors of the *Science* paper (who included founding directors of clinics and leading European institutions in occupational medicine) were based on ties to the asbestos industry are alarmingly unprofessional. My research on asbestos has been supported by federal grants (and never by the asbestos industry) for more than 15 years. I currently have grant support from the NIEHS, the National Heart, Lung and Blood Institute, and EPA. I have never participated as an expert witness on behalf of any party in asbestos-associated litigation, nor have I served as a paid consultant to the asbestos industry in related matters.

The *Science* paper was extensively peer-

reviewed by several scientists and supported by a subsequent editorial (19). Approximately a dozen letters, none challenging the factual basis of the data we presented with convincing and new peer-reviewed papers, were received. Hence, there was understandably no press coverage of these letters. Unsurprisingly, the vast majority of letters received, including those printed, were repetitious (i.e., citing that chrysotile asbestos causes disease in rats at astronomical concentrations, as do a variety of nuisance dusts), political as opposed to scientific in nature, and submitted by individuals associated with labor unions or employed by the plaintiff bar (7). Contrary to Rall's statement, "overwhelming evidence from current research" to support their views was lacking.

Why the attack on our *Science* paper more than 4 years since its publication? Obviously, it's more than a matter of science. Put candidly, our paper and supporting ones not only changed the tide of press coverage on asbestos from panic to a perspective on occupational versus environmental risks of asbestos, but also curbed multibillion dollar, asbestos-related property damage litigation as well as a burgeoning asbestos removal industry. In combination with the HEI-AR panel report, our paper also influenced EPA policy to drop consideration of mandatory asbestos surveillance programs in public buildings and focus on management of asbestos in place as opposed to rampant removal (20). Although scientifically merited, these events have led to an unsuccessful counter-campaign fueled by the plaintiff bar, the asbestos removal industry, and labor unions.

In the interest of scientific professionalism, opponents should focus more on obtaining scientific data to support their contentions and less on innuendos, witch hunts, and smear tactics. Allegations of conflicts of interests by scientists with opposing views should be put into perspective with their own biases and activities. It is time to work together to continue preventive medicine and to develop therapeutic measures for asbestos-related diseases as needed by workers.

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The Question of Balance

In a recent editorial, "Media and Science: Harmless Dioxin, Benign CFCs, and Good Asbestos" (*EHP* 102:10), David P. Rall decries the "mistakes in editorial policy and reporting" in science and medicine, and suggests "balancing controversial views in the same issue and to invite letters and commentary for publication in the same issue" of the journal in which the material is published. He dealt with "serious environmental concerns: dioxin, chlorofluorocarbons (CFCs), and asbestos."

In regard to the dioxin story in particular, Rall quoted from the Fingerhut (*J*) report that "workers exposed to dioxin for more than 2 years and observed for at least 20 years had a 46% greater cancer death rate than expected." This article was accompanied by an editorial which many newspapers quoted at the time of the report, and others did not, as Rall chose not to. The editorial, written by Bailar (*2*), notes that "Results are again equivocal. Parties on both sides of the continuing debate about the regulation of dioxin exposure will no doubt cite this work in support of their positions" (as I am doing, and as Rall did, by not citing the editorial). Bailar continues:

Some cancers were indeed more frequent in an exposed group than among controls, but the differences were for the most part not statistically significant, and the exceptions might be explained by a combination of small, unavoidable biases in the data and the multiple post hoc comparisons. (Examine enough data at the usual 5 percent level of significance and about 1 time in 20 you will find a statistically significant result where there is no real effect.)

The information is there, but depending on the reporter, the newspaper, or the scientist you will inevitably get a different story. Following the Fingerhut article, for example, one newspaper headline read "Chronic Dioxin Exposure ups Cancer Risk," another read "More Research into Dioxin Urged," and still another "The Deadliness of Dioxin Put in Doubt By New Data."

Rall also states that "A 10-year follow-up of those exposed to dioxin after the chemical explosion at Seveso in 1976, published in *Epidemiology* this summer, showed an increase in some cancers" (*3*). I believe Rall might have mentioned, for completeness sake, that the report also indicated that in one group of exposed

individuals, there was a decrease in breast and uterine cancer, as was observed in a very balanced news report from the *New York Times* on 26 October 1993, by Keith Schneider.

More recently, the article by Davis et al. "Decreasing Cardiovascular Disease and Increasing Cancer among Whites in the United States from 1973 through 1987" in the *Journal of the American Medical Association* (*4*), was accompanied by an editorial (*5*) which was in part critical of the work, yet many media reports failed to recognize the criticism of the editorial, while others gave a very balanced report by using both the article and the editorial (in particular Jane Brody of the *New York Times*, 16 February 1994).

These examples indicate that scientific information is readily available, either in the publication itself, or in the now common practice of the concurrent editorial comment, and is more often critical of the publication than not. Concurrent letters to the editors are not necessary. Reporters, in my opinion, have a good understanding of what they read; it is what they choose to report that may be faulted.

For the various media, the old saying applies, namely good news is no news, bad news is good news, except, of course, if you have a bias, and bias is not limited to reporters. We scientists are full of it, too.

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Regarding Bias

I was saddened to read Rall's editorial comment on the paper in *Science* entitled "Asbestos: Scientific Developments and implications for Public Policy" (*1*). He suggested the "industry association" of